not indicated; if high pressure is present they may be tried cautiously. Digitalis is not indicated for the aortic condition alone and may do harm. For the attacks of pain—if severe enough to demand treatment—codeine may be given by the mouth; morphine is rarely required, but should be given if the pain is very severe. Some patients, even with a low blood pressure, find that nitroglycerin ($\frac{1}{100}$ to $\frac{1}{50}$ grain) gives almost immediate relief, and there can be no objection to its use. Others find relief from a dose of aromatic spirit of ammonia (ʒi) with spirit of chloroform (♏xv).

In general, it may be said that a quiet even life, with avoidance of physical and mental strain, a diet limited especially in amount, open bowels, and meeting special indications when necessary, are the main points. With these we may hope to give the patients in whom the heart is not markedly affected many years of comfortable life. When cardiac breakdown appears, it must receive proper treatment.

CONCLUSIONS. 1. Dilatation of the aorta, considered apart from aneurysm, is relatively common and probably occurs more frequently than aneurysm.

2. Two main etiological factors stand out—(*a*) acute infections, notably rheumatic fever, and (*b*) those which cause general sclerotic changes.

3. The symptoms often suggest disease of the heart itself.

4. The physical signs are often very definite, of which visible pulsation in the upper interspaces, dulness over the upper sternum and adjoining interspaces, and the fluoroscopic examination are the most important.

5. Pressure signs are comparatively common, but are rarely severe.

6. The diagnosis can usually be readily made from aneurysm.

7. The outlook as regards dilatation is good.

HEART BLOCK WITH AN INDICATION OF GENUINE HEMISYSTOLE.[1]

BY ALFRED STENGEL, M.D.,
PROFESSOR OF CLINICAL MEDICINE IN THE UNIVERSITY OF PENNSYLVANIA,
AND
WILLIAM PEPPER, M.D.,
ASSISTANT PROFESSOR OF CLINICAL PATHOLOGY IN THE UNIVERSITY OF PENNSYLVANIA,
PHILADELPHIA.
(From the William Pepper Laboratory of Clinical Medicine.)

THE case of heart block which furnished the basis for the observations which we wish to report was not of such exceptional character that a separate publication of the clinical features would be of value; but the numerous tracings which were obtained furnished some interesting information and by lucky chance one of these proved of unusual importance in throwing some light on the possibility of true hemisystole. We shall give only the outlines of the history and clinical aspects of the case and then proceed to the discussion of the more interesting features.

---

[1] Read at a meeting of the Association of American Physicians, Washington, D. C., May 3 and 4, 1910.

The patient was an Italian, aged thirty-nine years, a cabinet maker, and married. Three years ago pains in the epigastrium occurred. One year ago he had a severe attack of the same pain with an attack of syncope, but noticed nothing wrong with his heart. He had a fainting attack in August, 1908. Ten days before admission to the University Hospital he had epigastric pain, and was in bed for a few days. On returning to work the pain recurred at once, and he then noticed that it came with each beat of the heart and that the heart was beating very slowly. He went home, where he had several attacks of faintness, vertigo, and vomiting.

When twenty-three years old he had a suppurating bubo and a sore on the penis. No secondaries followed, but after leaving the hospital where he went for treatment several of his teeth fell out. He has four living healthy children; his wife had one miscarriage before the four children were born. He has always been a healthy man, although he has not felt quite so well during the last three years. He has some pain along the edge of the tibiæ, especially in winter. The Wassermann reaction was positive.

The heart was enlarged one and one-half inches to the right of the sternum, but not beyond the left midclavicular line. A long systolic murmur was heard, and a dull second sound. When admitted, on February 5, 1910, at 6.30 P.M., the pulse rate was 62. The next morning it was 76, but it rapidly fell to 22 by noon, and did not rise above 34 until 6 A.M. on February 9.

The features that we wish to discuss are the following:

1. That although for a time complete block existed and later partial heart block, during the patient's stay in the hospital no Stokes-Adams syndrome was present.

2. That the complete block, after the administration of atropine, promptly changed to an incomplete block with a 3 to 1 rhythm, then to a 2 to 1 rhythm, and later to a normal rhythm.

3. That the 2 to 1 rhythm and the normal rhythm for a time alternated one with the other, and a number of tracings were obtained showing this change while the tracings were actually being made.

4. That it furnished indications of genuine hemisystole during the transition of the normal rhythm to a 2 to 1 rhythm.

5. That from this same tracing it could be demonstrated that the origin of the *c* wave is the right ventricular contraction, rather than the carotid pulsation.

6. That the *a-c* interval is longer in such cases as this during normal rhythm, while it may be normal in length during partial block.

7. That the auricular rate becomes faster when the normal rhythm changes to a 2 to 1 rhythm, and slower when the opposite change occurs.

8. That explanations were suggested for the increased or decreased auricular rate during block and normal rhythm and for the irregular ventricular rhythm during complete block.

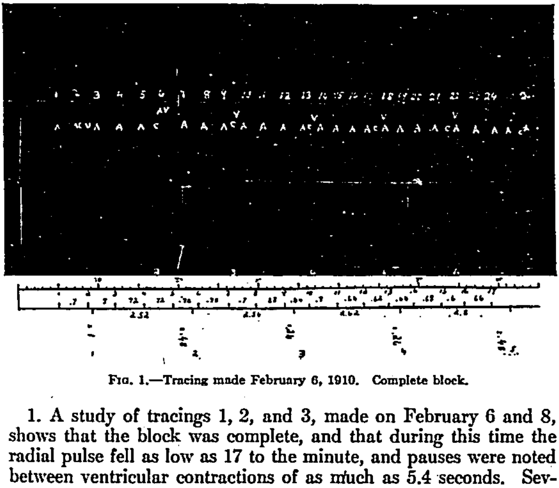

FIG. 1.—Tracing made February 6, 1910. Complete block.

1. A study of tracings 1, 2, and 3, made on February 6 and 8, shows that the block was complete, and that during this time the radial pulse fell as low as 17 to the minute, and pauses were noted between ventricular contractions of as much as 5.4 seconds. Several times 7 auricular contractions were observed between 2 ventricular ones, and yet not the slightest evidence of any Stokes-Adams syndrome was noted during the patient's stay in the hospital. During the phase of complete block the average ventricular rate from 35 measurements taken from 8 different tracings was 19.2 per minute, while the average auricular rate from 174 measurements was 80.7 per minute, a little more than four times as many to the minute.

2. A study of tracings 2 and 3, made at 1.30 P.M. on February 8, shows a complete block at that hour, and tracing 4, made 4 hours later after two hypodermic injections of atropine had been given —one at 1.30, of $\frac{1}{100}$ grain, and another at 4.30 of $\frac{1}{75}$ grain— shows that the complete block has changed to an incomplete one with a 3 to 1 rhythm. In tracing 4 the auricle was beating about

81 times per minute and the ventricle 27. Atropine was then given by the mouth every third hour, $\frac{1}{200}$ grain for two days, then withheld for four days, and then given again for a week. Tracings on the ninth day after the change from complete block to incomplete block, with a 3 to 1 ryhthm, showed a 2 to 1 rhythm, and thereafter up to the present date the tracings have shown either a 2 to 1 rhythm or no block at all. He has apparently had no return of the complete block since the two hypodermic injections of atropine on February 8.

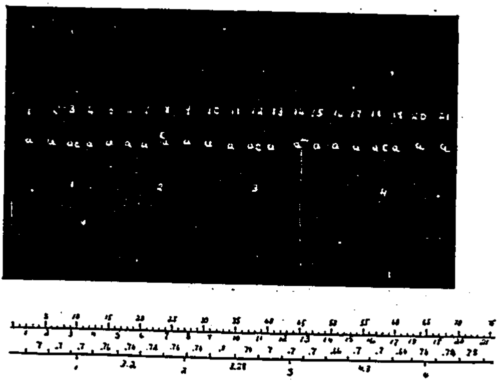

FIG. 2.—February 8, 1910. Before atropine was given. Complete block. Irregular ventricular action.

Figs. 3, 4 and 5 can be well considered together. On examining tracing 5 it will be observed that the normal rhythm changes suddenly to a 2 to 1 rhythm, brought on possibly by a deep breath that the patient had taken about seven seconds before, though more probably coming on spontaneously, as at this time the 2 to 1 rhythm and the normal rhythms frequently alternated. It will be seen that the first six *a* waves are followed, as they should be, by a *c* wave and accompanied by a wave in the radial tracing, but that the seventh *a* wave is not followed by a *c* wave, although accompanied in the radial by a wave exactly like the preceding one. The eighth *a* wave is followed by a *c* wave and accompanied by a wave in the radial, the ninth *a* wave is not followed by a *c* wave and is not accompanied by a wave in the radial, and from here on the 2 to 1 rhythm which has been started persists. Now, how are we to explain why the seventh *a* wave is not followed by a *c* wave, although accompanied by a wave in the radial tracing? There being an *a* wave shows that the right auricle contracted, and there being a wave in the radial shows that the left ventricle contracted following the auricular contraction, but why is there no *c* wave? If the *c* wave is caused largely by the carotid

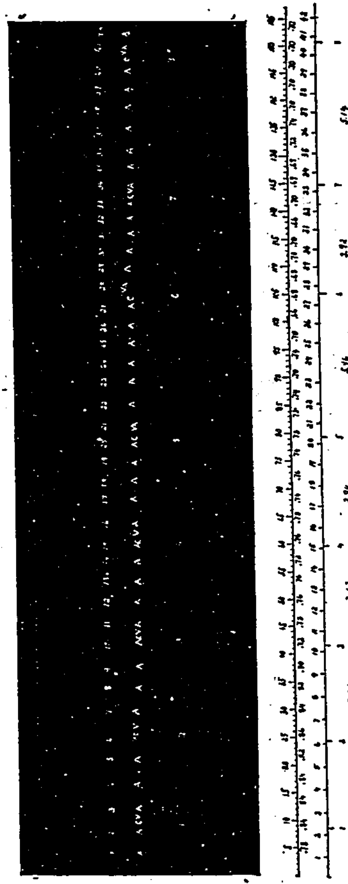

FIG. 3.—February 8, 1910. Before atropine. Complete block. Irregular ventricular action.

artery, we should here in this case have a *c* wave, because, judging from the radial, the left ventricle contracted with its customary strength and at the proper time. Therefore, it seems to us that the *c* wave cannot be due to the carotid, but must be due to the right ventricle, and that in this particular instance the right ventricle did not contract or contracted feebly, although the left did contract normally. Coming as this did just before the institution of the 2 to 1 rhythm, it certainly suggests that the right ventricle ceased first, and then the left ventricle followed suit, and that then a perfect 2 to 1 rhythm resulted. This would seem to prove that one ventricle can contract normally while at the same time the other ventricle either contracts very feebly or not at all. Probably it would be unwise from this single observation to say that in this particular case the division of the bundle of His going to the right ventricle was more involved or damaged than the division going to the left ventricle, but if further similar findings had been noted, possibly such a localization could have been made.

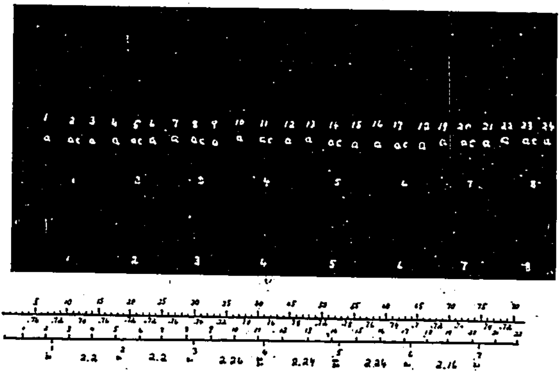

FIG. 4.—February 8, 1910. After atropine had been given. Incomplete block with 3 to 1 rhythm. *a-c* interval usually normal.

The earliest reference to hemisystole we could find was in an article by Charcelay in 1838, who wrote a memoir on some remarkable cases of asynchronous contraction of the ventricles of the heart. Then in 1868 von Leyden[2] reported the first of his series of cases, the last of which appeared in 1908, forty years later. These cases of von Leyden's have been considered by most other writers as

---

[2] Virchow's Archiv, 1868.

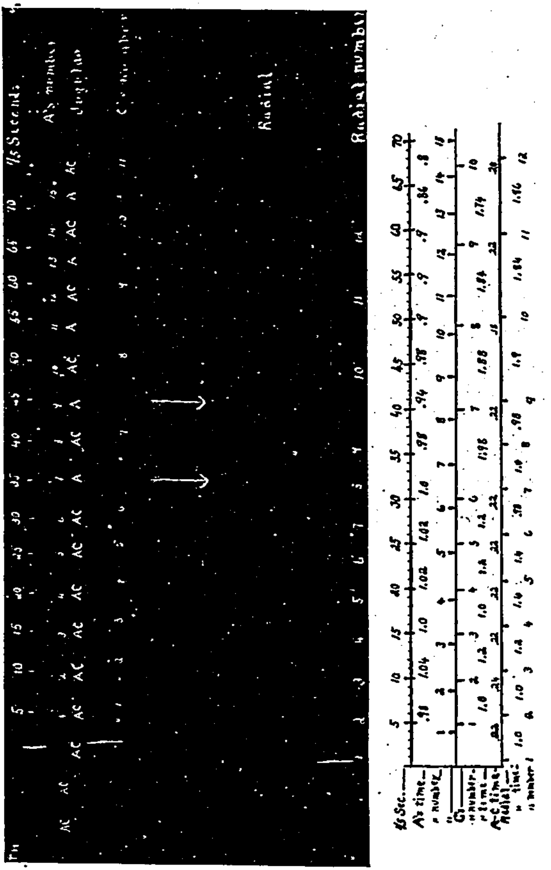

FIG. 5.—February 17, 1910. Change from the normal rhythm to a 2 to 1 rhythm. Note that the seventh *a* wave is *not* followed by a *c* wave although it *is* accompanied by a wave in the radial (No. 8), and that the ninth *a* wave is *not* followed by a *c* wave and is *not* accompanied by a wave in the radial.

typical examples of pulsus bigeminus. Hering, in particular, has been very insistent upon this point, and thinks that the term hemisystole should be abolished, because although on inspecting the hearts of dying animals one can observe one ventricle beating after the other has ceased to beat, or one ventricle contracting more rapidly than its fellow, such contractions as these can only be noted in dying hearts, and are not, he thinks, compatible with life.

Barker and Hirschfelder,[3] in a very important paper entitled "The Effects of Cutting the Branch of the His Bundle going to the Left Ventricle," reported that they found, when they cut the left branch without injuring the right, that complete block resulted, but both ventricles contracted synchronously. In one experiment no block resulted, and they, therefore, conclude that the bundle of His plays little if any role in the coördination of the two ventricles.

Eppinger and Rothberger[4] report some studies of the consequences of section of Tawara's branches of the conducting apparatus. Their observations of the electrocardiograms they obtained showed after section of the left branch a curve significant of right-sided extrasystoles, and on section of the right branch, left-sided extrasystoles. When the ventricular contractions are infrequent, the auricular waves can be seen before the ventricular. The authors conclude from their studies that the stimulus originating in the auricles passes down through the bundle of His to its division and then to the left and right ventricles through the branches. When the left branch is cut, the impulse does not flow to the left ventricle, and it is, therefore, not stimulated to direct contraction; the right ventricle contracts first and the left receives its impulse in an indirect way through the right. This state of affairs is the same as occurs when in case of vagus inhibition the right ventricle is stimulated from its wall and consequently contracts before the left. The authors obtained somewhat similar results in a previous study in which cardiac lesions were produced by injections of silver nitrate solution.[5] They refer to the fact that Aschoff had raised the question in connection with his pathological studies whether a lesion of one of the Tawara branches or its division would not impair cardiac action, and Monckeberg has shown that ecchymotic hemorrhage and subendocardial fatty metamorphosis is apt to occur in the position of the conducting bundle and its branches. The authors finally call attention to the fact that in their operations on dogs, section of one Tawara branch was followed by gallop-rhythm characterized by a disturbance of synchronous contraction of the ventricles. In the Medical Clinic of Vienna, they recently observed two fatal cases in which during life the form of the electrocardiogram had suggested a diagnosis of interruption of conduction to one ventricle. The autopsies showed macroscopically a large myocarditic lesion in the septum. How far this may have invaded the conducting tissues will have to be determined by careful microscopic examination.

---

[3] Trans. Assoc. Amer. Phys., 1909.

[4] Wien. klin. Woch., 1909, No. 31.

[5] Ztsch. f. klin. Med., lxx, 1.

Pulsus bigeminus simulates hemisystole because the second or smaller beat, which is an extrasystole, although giving a distinct wave in tracings made from the apex and in the neck does not give any wave in the radial. McKenzie has always insisted that the *c* wave was caused by the carotid, and this view has been adopted by some and rejected by others. It is our belief that the *c* wave can be caused by the carotid artery if the recorder be placed directly over this vessel, but we also believe that by moving the recorder a venous wave can be obtained that has nothing to do with the carotid but comes from the right ventricle. In other positions both these factors undoubtedly may take part in the formation of the so-called *c* wave.

6. A study of a number of the tracings showed that when the rhythm was normal the *a–c* interval averaged 0.2208 second, this figure being taken from 60 measurements; when the rhythm was 2 to 1 the *a–c* interval averaged 0.205 second, as estimated from 24 measurements; when the rhythm was 3 to 1 the same figure was obtained from the average of 7 measurements. Greater variations were noticed in the length of the *a–c* interval during normal rhythm than at other times. No evidence of progressive increase in the length of the *a–c* interval leading up to a partial block was found at any time.

7. That the auricular rate is slower when normal rhythm exists and faster when a 2 to 1 rhythm has come on, is found to be true from a series of measurements taken from a number of tracings. These changes in rate come on immediately after the change one way or other sets in, but we find that there is a greater immediate disturbance of the auricular rate of contraction when the rhythm changes from a 2 to 1 to a normal rhythm than when the normal changes to a 2 to 1 rhythm. Thus, we find an average drop of some 17.9 beats per minute in the rate or a change from 74.2 to 56.3 when there was a restoration to normal rhythm; while when the normal rhythm changed to a 2 to 1 rhythm there was an increase in the auricular rate of but 10.6 beats in the minute or a change from 59.7 to 70.3. These averages were obtained by measuring only about 20 beats immediately following the change in rhythm.

8. In attempting to determine the cause of greater auricular rate during 2 to 1 or 3 to 1 rhythm than when no dissociation existed, it has occurred to us as reasonable that the interference with auricular outflow, which is present at each alternate or third auricular contraction, would necessarily leave the auricle relatively unemptied and would thus occasion an earlier stimulus to contraction, which would result in a more rapid rate. When a change from normal to

2 to 1 rhythm occurs the quickening of auricular rate is less prompt than is the slowing when a 2 to 1 rhythm changes to the normal. An explanation for this circumstance may be found in the fact that resumption of normal rhythm after a 2 to 1 rhythm at once relieves the auricle of back pressure and the consequent stimulus to increased rate. Such a sudden relief is followed by prompt re-adjustment of auricular activity, whereas the effect of change from normal to a 2 to 1 rhythm and the consequent gradual overfilling of ventricle and auricle would be met by a slower response.

In this case during the time of complete block the ventricular rhythm was not at all times regular, as has been already mentioned, although the ventricle usually contracted about 20 times to the minute, or about every three seconds; at other times there were irregular pauses, the longest being 5.4 seconds. There were, however, no extrasystoles at any time. It is known from physiological experiment that, owing to poorly developed or retained independent rhythmicity, a portion of the ventricle, as, for example, the severed apex of the ventricle, tends to cease contracting after a time and will resume regular contractions only after an external stimulation. It is, therefore, possible that the continued action of the ventricle sometimes for long periods and despite occasional long pauses in cases of complete block may be due to the stimulus of distention with blood resulting from the continued auricular action.

THE TREATMENT OF ANEURYSM OF THE AORTA BY THE INTRODUCTION OF WIRE AND THE PASSAGE OF A GALVANIC CURRENT.[1]

By Augustus A. Eshner, M.D.,

PROFESSOR OF CLINICAL MEDICINE IN THE PHILADELPHIA POLYCLINIC AND COLLEGE FOR GRADUATES IN MEDICINE; PHYSICIAN TO THE PHILADELPHIA GENERAL HOSPITAL; ASSISTANT PHYSICIAN TO THE PHILADELPHIA ORTHOPEDIC HOSPITAL AND INFIRMARY FOR NERVOUS DISEASES; CONSULTING PHYSICIAN TO THE MERCY HOSPITAL, PHILADELPHIA.

Aneurysm of the aorta is essentially a fatal disorder sooner or later, partly on account of the resulting interference with the blood distribution and pressure phenomena, but largely and inherently because of the underlying degenerative state of the vessel wall, so that if correction or repair be effected in one situation, active manifestations are likely to appear in another situation. The affection is almost invariably associated with more or less widespread atheroma of the arterial system, and antecedent syphilis appears to be far and away the commonest cause. The dilatation or diverticulation of the bloodvessel must, accordingly, be looked upon merely as a local expression of changes widely distributed throughout the vascular system, and accordingly the results of any therapeutic procedure can at best be only palliative, except in the small group of cases in which the lesion arises from purely local influences, such as traumatism or adjacent disease. Nevertheless, it will not be gainsaid that measures of temporary relief are often indicated, either for the amelioration of symptoms or for the prolongation of life. In this connection the fact should not be ignored that under certain conditions life may be continued for many years in the presence of an aneurysm and also that a sacculated aneurysm of the aorta may, and occasionally does, undergo spontaneous cure by the total occlusion of the aneurysmal sac by fibrous laminated adherent clot, and be discovered at autopsy only as an incidental phenomenon. Furthermore, it is impossible to foresee the outcome of the brilliant experimental work that has been done and is being continued in the plastic surgery of the vascular system. Nevertheless, there will remain cases in which symptomatic treatment only will be possible.

---

[1] Read at a meeting of the Association of American Physicians, Washington, D. C., May 4, 1910.

In the treatment of aneurysm of peripheral vessels, with abundant anastomotic connections, deligation, with or without excision or resection of the aneurysmal sac, has yielded satisfactory and at times even brilliant results, but this mode of procedure is scarcely applicable to aneurysm of the aorta, and more particularly, its thoracic division, partly on account of anatomical limitations with respect to the establishment of a collateral circulation, and partly by reason of the practical inaccessibility of the disease focus. Various modes of treatment, dietetic, medicinal, physical, have been applied to aneurysms of the latter class, with varying measure of success. Among the most rational and most useful of these may be mentioned rest, restricted diet, ergot, iodides, calcium chloride, adrenaline, introduction of foreign bodies, electricity. Of foreign bodies there have been introduced into the aneurysmal sac substances such as horsehair, catgut, silk, watchspring, needles, and wire of various kind.

Charles H. Moore and Charles Murchison[2] first employed fine iron wire in this manner, introducing twenty-six yards through a cannula into an aneurysm of the thoracic aorta. The tumor was at first reduced in size, but subsequently it became larger, and the patient died on the fifth day. Postmortem examination disclosed abscess of the left kidney and pericarditis.

The possibility of treating aneurysm by transfixion with needles occurred to Velpeau[3] and to Benjamin Phillips;[4] while Ciniselli[5]

---

[2] Medico-Chirurgical Transactions, 1864, xlvii, 129.

[3] Memoires de l'Academie des Sciences, December, 1830.

Medical Gazette, 1831, 499; also pamphlet, A Series of Experiments for the Purpose of Showing that Arteries may be Obliterated without Ligature, Compression, or the Knife, 1832.

[5] Gazette des Hôpitaux, 1868.